

# On the relationship between molecular weight and anticoagulant activity of heparins : in vitro and ex vivo studies

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## ***STELLINGEN - POSITIONS***

1. **Variation of the specific antithrombin activity of monodisperse heparin fractions is due to variations of their content of AT III binding sites.**
2. **The serine-protease factor VIIa is not inhibited by antithrombin III.**
3. **The onset of thrombin in platelet rich plasma is due to heparin fractions with MW  $\geq 5,400$  (ACLM); the decrease of the peak is due to fractions with MW  $< 5,400$  (BCLM).**
4. **We could surmise that ACLM might prevent thrombosis, whereas BCLM cures it.**
5. **The U.S.P. unit for the specific activity of heparins does not fulfil its purpose as soon as low molecular weight heparins are to be characterised. It should be replaced by a unit in the I.S.**
6. **Factor VII possesses no intrinsic proteolytic activity toward factor X or factor IX. (Wildgoose et al. Biochemistry 29; 3413-3420; 1990).**
7. **Cholera is an attribute of poverty (Nature, 350, 6320: 640; 1991).**
8. **The disappearance of the Amazonian forest might entail the loss of drugs potentially crucial for future medicine.**
9. **Chagas's disease (Trypanosomiasis cruzi) will dwindle together with the straw roof housing.**
11. **In pharmacological studies, it is generally accepted that the concentration of a drug in circulation is determined by its input and rate of clearance. Somehow this fact is overlooked in studies of circulating proteins.**
12. **In the "Low Countries", Holland is not a synonymous of The Netherlands.**

*Vicky Bendetowicz  
Maastricht, 27<sup>th</sup> March 1992*